dine bases in urine and tissues is not proved.

The main evidence for belief in presence of a toxic compound in the blood of animals in tetany after parathyroidectomy seems to lie in the observation that if such animals be bled, and then a corresponding amount of normal saline be injected, there is temporary relief, due, according to the protagonists of the guanidine theory, to dilution of the toxic compound. But Greenwald, testing the blood from such animals actually in tetany on normal animals, could find no evidence of toxicity, while recently Swingle has shown that the true cause of the actual relief that really does occur is a temporary rise of the blood calcium. Collip's work, definitely associating the secretion of the parathyroids with maintenance of the normal level of blood calcium, dealt a severe blow to the guanidine theory, and Collip and Clark have now shown that the chemical blood picture of dogs in tetany after parathyroid removal and after guanidine injection is sufficiently different to lead to the conclusion that the guanidine intoxication theory of parathyroid tetany is incorrect.

Most, if not all of the parathyroid extracts on the market, with the exception of those prepared by Collip's procedure, are inactive. Last year, White and Cameron showed that Collip's active extract is inactive when tested by Vines' guanidine procedure, according to which on the other hand, nucleic acid, present, in all fresh and dessicated tissues, is more active than dessicated parathyroid preparations.

Wherever the evidence for the guanidine series of theories has been tested it has been found to be either invalid or insufficient. The theory associating guanidine detoxication with parathyroid function we may discard. Major's theory that guanidine is associated with arterial hypertension requires much more rigid proof than has yet been offered. The days of the guanidine heresy seem almost over.

A. T. CAMERON

INHERITANCE IN CANCER: A NOTE ON THE WORK OF MAUD SLYE

THERE appeared recently an important article* dealing with the principles of inheritance in general, and the application of these to the heredity of cancer. The author, Professor Maud Slye, has devoted long years of patient and careful study to the great problem of malignant disease and, in her laboratories in Chicago, has conducted many thousands of experiments upon cancer in mice. She now demonstrates clearly the remarkable and extremely important fact that, in this animal at least, spontaneous cancer is inherited, and arises through the action of a recessive factor for abnormal tissue growth. In order, however, that such recessive characters may be expressed in an individual they must be present in double quantity, that is, they must be represented in *both* parents; for, if occurring in only single quantity they remain latent, although they may be transmitted to the offspring.

The mode of inheritance of cancer is graphically and convincingly brought out in a series of charts which serve to demonstrate the astonishing fact that an individual may inherit cancer from a father and mother who are quite free from the disease, and in whose family history there is no carcinomatous taint, but who both carry the factor for cancer latent. The factor for cancer may thus remain lurking in the germ plasm without being accompanied by any actual disease in the individual, and may never exhibit its potentiality in the production of the malady unless mated with another simi-

* Slye, Maud, Some misconceptions regarding the relation of heredity to cancer and other diseases: Studies in the incidence and inheritability of spontaneous cancer in mice, *Jour. Am. Med. Ass.*, 1926, lxxxvi, 1599-1606.

lar factor in another individual. With no other artificial measures other than the choice of appropriate mates (those known to carry the recessive factor for abnormal tissue growth), she can change what has been for generations an apparently cancer-free strain of mice into a strain in which every mouse develops the disease.

Dr. Slye thus accounts for the sporadic cases of cancer in the human subject, where no hereditary tendencies are discernible, and her work seems to bring into harmony with the inheritance theory of cancer, a circumstance which up to the present has been very puzzling. This discussion is quite apart from the cases of cancer where there is a definite family history of the disease. Members of such families, of course, are more apt to develop carcinoma than are those from families in which it has never made itself manifest.

Obviously, experimental work of the type which Miss Slye has carried out demands remarkable skill and infinite patience. The author emphasizes the necessity for a very careful study of the animals used in genetic experimentation, by exhaustive breeding tests, to demonstrate whether or not they are completely lacking in any factor for abnormal tissue growth. By such means it is possible to select cancer-free strains, just as it is possible to select those which have a recessive cancer-producing factor. Applying the same principles to the human subject, it should be possible, if the conditions could be controlled, to develop only a cancer-free human stock, and to eliminate this dread scourge altogether from the human race. That is one of our eugenical problems.

Atention is called by Miss Slye also to the fact that the terms "congenital" and "hereditary" are often confused, as, for instance, in connection with congenital syphilis. Hereditary defects are inherent in the fertilized ovum, and have been present in the individual's progenitors, either latent or evident. A hereditary defect may be manifest at birth or may lie dormant for many years before it is apparent, as in the case of peroneal atrophy. Congenital defects, on the other hand, arise through extraneous influences, such as those of a chemical or bacterial nature, exerted upon the developing embryo, and are in no manner to be referred to defects in the germ cells. The tendency to designate as congenital what is really hereditary militates against a clear knowledge of the importance of heredity in the transmission of disease.

MADGE THURLOW MACKLIN

THE PSYCHOGENETIC ELEMENT IN EPILEPSY

OCCASIONALLY one has the opportunity of witnessing an hysterical attack in a patient afflicted with essential or idiopathic epilepsy, and again one may see a typical major epileptic seizure in a patient whose make-up and behaviour are distinctly hysterical.

We are on safe ground when we assume that in many cases, labelled epilepsy, a psychogenetic element is present. If, as is well known, an emotional disturbance such as anger or excitement may bring on a seizure, are we unreasonable in formulating an hypothesis of suppressed or repressed emotional conflicts constituting in some cases the origin of epileptic attacks, the latter being simply an expression of the former? Truly this will hardly apply to the epilepsy of infancy, but it might well apply to the epilepsy of childhood and adult life.

The line between epileptic and hysterical symptoms is sometimes indistinct. Attacks of dizziness, for example, may be diagnosed as *petit mal* or hysterical according to the bent of the examiner. Amnesia with regard to a convulsive outbreak might point in either direction.

Loss of consciousness is no longer considered indispensable to the diagnosis of petit mal, and bed-wetting and biting of